studied 100 hypertensive patients, without a history of prior myocardial infarction, matched for age, race, and gender. The QT interval was measured from the onset of the QRS complex to the end of the T wave in each lead of a standard 12-lead ECG. QT dispersion was calculated as the difference between the longest and shortest QT intervals (QT max-min). LV mass was measured by 2-D guided M-mode echocardiography. LVH was defined as LV mass index $\geq$ 134 gm/m^2 in men and $\geq$ 110 gm/m^2 in women. Fifty patients had echocardiographically documented LVH and 50 had normal LV mass index. The ECG measurements were made by an investigator blinded to the echocardiographic results.

Patients with and without LVH were similar in age (55 $\pm$ 14 vs. 53 $\pm$ 12 years, p = NS), race (66% African American, 34% Caucasian), and gender (58% female). The mean LV mass index was 169 $\pm$ 54 gm/m^2 in patients with LVH and 95 $\pm$ 19 gm/m^2 in those with normal LV mass ($p < 0.001$ for difference). Patients with LVH had a greater mean QT max-min (70 $\pm$ 27 msec) than hypertensives without LVH (46 $\pm$ 27 msec, $p < 0.001$ for difference). The association between LVH and increased QT dispersion remained significant when men, women, African Americans, and Caucasians were examined separately.

We conclude that LVH due to hypertension is associated with increased QT dispersion. These findings suggest a possible role for repolarization inhomogeneity in the enhanced risk of sudden cardiac death in patients with hypertensive LVH.

1030-46 Prospective Study of Prognostic Value of Arrhythmogenic Markers In Systemic Hypertension

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Hypertensive left ventricular hypertrophy (LVH) is associated with increased risk of arrhythmias and mortality. However, no clinical study demonstrated a significant relation between ventricular arrhythmias or other arrhythmogenic markers and mortality in systemic hypertension.

To evaluate the prognostic value of arrhythmogenic markers in systemic hypertension, we included between 1987 and 1993 214 hypertensive patients (pts), 127 men and 87 women, 59.1 $\pm$ 12.8 years old, without symptomatic coronary disease, myocardial infarction, systolic dysfunction, electrolyte disturbances or antiarrhythmic therapy. At inclusion, an ECG, a 24 h Holter ECG (204 pts) with Lown classification of ventricular arrhythmias, an echocardiography (reliable in 187 pts) with left ventricular mass index and ejection fraction calculation, a SAECG (125 pts, enrolled after 1988) with ventricular late potentials (LP) were recorded. QT interval dispersion (QTd) was calculated on 12 leads standard ECG and LVH was appreciated.

At baseline echocardiographic LVH was recorded in 63 pts (33.7%) with normal ejection fraction (75 $\pm$ 7.4%). Advanced Lown classes were found in 71 pts (34.8%), and LP in 27 pts (21.6%). After a mean followup of 42.4 $\pm$ 26.8 months, all-cause mortality was 11.2% (24 pts); 17 pts died of cardiac causes (7.9%); of these 9 patients (4.2%) died suddenly. In univariate analysis, age, strain pattern of LVH, advanced Lown classes and abnormal QT dispersion (> 80 msec) were significantly related to global, cardiac and sudden death ($p \leq 0.01$). Left ventricular mass index was closely related to cardiac mortality (p = 0.002). LP failed to predict mortality. In multivariate analysis, age was an independent predictor of global mortality, cardiac and sudden death. Advanced Lown classes was an independent predictor of global mortality, increasing the risk of death 2.9 fold [1.2–6.8] (CI 95%). Abnormal QT dispersion was an independent predictor of cardiac death, increasing the risk of cardiac death 3.2 fold [1.1–9.6] (CI 95%). The association of abnormal QT dispersion and advanced Lown classes was an independent predictor of global and cardiac mortality, increasing the risk of death 2.9 fold [1.3–6.6] and the risk of cardiac death 3.1 fold [1.1–8.8] (CI 95%).

Thus, in hypertensive pts an abnormal QT dispersion and an advanced Lown class, but not LP, have a prognostic value.

1030-47 Ventricular Remodeling During Reversal of Left Ventricular Hypertrophy in Arterial Hypertension

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Antihypertensive drugs are known to reduce ventricular hypertrophy. To investigate the process of ventricular remodeling during the course of such a reduction, we studied 25 patients with untreated essential hypertension. Patients received 25 mg captopril twice a day for 24 months. Cardiovascular and hemodynamic findings were assessed by echocardiography at baseline and after 6, 12, 18 and 24 months. Blood pressure was reduced from 165 $\pm$ 17/102 $\pm$ 5 mm Hg to 127 $\pm$ 14/89 $\pm$ 7 mm Hg ($p < 0.001$) after 6 months and remained at those levels thereafter. Left ventricular mass index was reduced 6%, 24%, 34%, and 41%, and interventricular septal thickness and posterior wall thickness were both reduced 8%, 17%, 25%, and 25% at 6, 12, 18 and 24 months, respectively (p = NS, < 0.01, < 0.001, and < 0.001, respectively). Left ventricular cavity area increased 15%, 26%, 39%, and 42%, and left ventricular wall area diminished 9%, 15%, 24%, and 30% at 6, 12, 18, and 24 months, respectively ($p < 0.01$, < 0.01, < 0.001, and < 0.001, respectively). Normalization of left ventricular mass index in all patients required 24 months of therapy.

These findings indicate that: 1) there is increase in cavity area which normalized wall stress preceding the reduction in left ventricular mass, 2) there is a delay for significant reduction in left ventricular mass index until after 18 months of therapy, and 3) there is progressive increase in cardiac index and reduction in total peripheral resistance even after 24 months of antihypertensive treatment.

1030-48 Effect of Chronic Bradykinin Infusion and Chronic Bradykinin-Receptor Blockade on Cardiac Hypertrophy in Angiotensin II-Treated Rats

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In previous studies, we demonstrated that in ANGII-treated rats, prevention of cardiac hypertrophy (CH) by enalapril was blunted by bradykinin (BK) blockade by Hoe140. The putative role of BK was assessed by chronic exogenous BK infusion and endogenous BK blockade in 54 male Sprague-Dawley rats infused with ANGII. ANGII (200 ng/kg/min) alone and associated with BK at low (BKa, 15 ng/kg/day) and high doses (BKb, 100 ng/kg/day) or with Hoe140 (Hoe, 300 μg/kg/day) were delivered by Alzet pumps for 10 days and compared to control animals (Veh). Values of mean arterial pressure (MAP, mmHg) and cardiac output (CO, ml/min/kg bw, microsphere technique) in conscious rats, and heart weight (HW, mg/g bw) at the end of the study are reported below. Results were submitted to ANOVA and expressed as M $\pm$ SEM.

	Veh	ANGII	BKa	BKb	Hoe
MAP	111 $\pm$ 2	133 $\pm$ 3*	130 $\pm$ 5*	140 $\pm$ 10*	137 $\pm$ 6*
CO	399 $\pm$ 37	233 $\pm$ 30*	302 $\pm$ 36*	383 $\pm$ 8#	276 $\pm$ 9*
HW	2.89 $\pm$ 0.05	3.56 $\pm$ 0.10*	3.59 $\pm$ 0.14*	3.59 $\pm$ 0.11*	3.55 $\pm$ 0.17*

*indicates $p < 0.05$ vs Veh; #$p < 0.05$ vs ANGII.

Thus, BK-receptor blockade did not influence the development of HTA and CH. High-dose BK increased CO mainly through an increase in stroke volume but did not affect final MAP and CH. In conclusion, in the absence of ACEI, BK do not seem to affect arterial pressure and CH in ANGII-treated rats.

1030-49 Is Long Term Evolution of Left Ventricular Filling Modified by Antihypertensive Treatment?

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The long term (> 3 years) evolution of left ventricular filling was assessed with pulsed Doppler recording of mitral flow in 73 never treated hypertensives. At entry we measured office BP, left ventricular mass (LVM, 2D guided M mode echo, Devereux's formula, indexed for height) and mitral flow (at the level of mitral annulus, measurement of peak velocities of early (E) and late (A) flows). At least 5 cycles of echo and Doppler were recorded on a strip chart recorder (100 mm/s). Fourteen pts (10 males, aged 47 $\pm$ 11 years, group 1) with borderline hypertension were left untreated, the others (46 males, aged 52 $\pm$ 10 years, group 2) received various non randomised drugs, mainly beta-blockers, during the whole follow-up. At the final visit (mean = 61 $\pm$ 13 months later) another echo-Doppler was performed in the same conditions. All tracings were coded and read blindly. ANOVA was performed with HR (measured on tracings) as a covariate for Doppler. In the whole population age ($r = -0.40$, $p < 0.0001$) and HR ($r = -0.38$, $p < 0.0001$) were correlated to E/A, not BP and LVM/h. In group 1, BP (153 $\pm$ 22/94 $\pm$ 14 Vs 154 $\pm$ 15/96 $\pm$ 10 mmHg) and LVM/h (131 $\pm$ 39 Vs 128 $\pm$ 34 g/m) remained unchanged, E decreased (53 $\pm$ 15 Vs 58 $\pm$ 12 cm/s, $p < 0.05$) with a trend to E/A decrease (0.94 $\pm$ 0.34 Vs 1.04 $\pm$ 0.34, p = 0.06). In group 2, despite a significant reduction in BP (146 $\pm$ 13/90 $\pm$ 9 Vs 168 $\pm$ 16/103 $\pm$ 8 mmHg, $p < 0.0001$), in LVM/h (141 $\pm$ 36 Vs 158 $\pm$ 38 g/m, $p < 0.001$) and HR (64 $\pm$ 10 Vs 72 $\pm$ 10 bt/min) we observed a significant decrease in E/A (0.87 $\pm$ 0.27 Vs 0.92 $\pm$ 0.29, $p < 0.01$). *Conclusion:* Peak velocity of early filling and E/A ratio decrease with age in hypertensives, treatment appears unable to slow this process despite BP lowering and LVH regression.